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Perioperative Myocardial Ischemia in Patients Undergoing Noncardiac Surgery—II: Incidence and Severity During the 1st Week After Surgery

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Because of the importance of postoperative myocardial ischemia and because substantial physiologic changes can occur for prolonged periods postoperatively, the incidence, severity and temporal course of myocardial ischemia were studied in 100 high risk patients during the 1st week after major noncardiac surgery. Electrocardiographic (ECG) changes consistent with ischemia were continuously monitored using ambulatory solid state ECG in the 100 patients with or at risk for coronary artery disease. Ischemic episodes were defined as reversible ST segment depression ≥1 mm or elevation ≥1 mm above the baseline value, with the baseline adjusted for respiratory and positional variation and temporal drift. All ischemic episodes were confirmed by three independent blinded investigators using hard-copy recordings. Total ECG monitoring time was 10,445 h.

Twenty-seven patients (27%) developed 437 episodes of ischemia during the 1st week after surgery. The total duration of ischemia was 18,658 min, or 1.8 min of ischemia/h monitored. Ischemia was most severe during the early (days 0 to 3) versus late (days 4 to 7) postoperative period: 284 versus 153 episodes; 2.2 versus 1.2 min of ischemia/h. The greatest severity occurred on postoperative day 3: 109 episodes, 3.4 min of ischemia/h monitored, 1.5 mm mean ST change and 130 min mean duration. However, in 8% of patients, severe episodes also occurred late: postoperative day 6 = 44 episodes, 1.7 min of ischemia/h monitored, 1.3 mm mean ST change (59% ≥ 2 mm) and 92 min mean duration. Most ischemic episodes (57%) were associated with tachycardia. Eighty-four percent of episodes were silent, unaccompanied by symptoms of angina, pulmonary congestion or syncope. All five severe adverse cardiac outcomes (unstable angina, myocardial infarction or cardiac death) were preceded by postoperative ischemia occurring ≥1 day before the outcome.

It is concluded that in at-risk patients undergoing noncardiac surgery: 1) postoperative ECG ST changes consistent with myocardial ischemia are most common during the 1st 3 days after surgery, with changes persisting for ≥1 week; 2) postoperative ischemia is clinically silent throughout the entire period and therefore difficult to detect; 3) postoperative ischemia may be related to the persistently elevated heart rate during the 1st week after surgery; and 4) an association between both early and late postoperative ischemia and severe cardiac outcomes is suggested.

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Recent data (1) suggest that postoperative myocardial ischemia is a major predictor of adverse cardiac outcome after noncardiac surgery. These findings imply that more intensive monitoring and aggressive therapy may be warranted during the postoperative period. However, the financial implications of such care may be prohibitive given the large number of at-risk patients (approximately 3 million annually in the United States) (2), the high incidence of postoperative ischemia (25% to 50%) (3–5) and the high cost of monitoring and care (6–8). Before pursuing such a costly course, we need to know more about the postoperative ischemia process, particularly its time course, duration, relation to myocardial oxygen supply and demand and, most importantly, its relation to outcome.

Our recent studies (1,9) demonstrated that of all periods, the postoperative period appears to be most important—early postoperative ischemia is both common and severe (9) and has a strong causal association with outcome. However, perioperative cardiac morbidity, including myocardial infarction, may occur later in the postoperative period, perhaps 3 to 7 days after surgery (2,10,11). If true, then *late* postoperative ischemia also may be prevalent and even a more powerful predictor of perioperative cardiac morbidity, indicating that more prolonged monitoring and therapy be instituted. We therefore determined the characteristics and

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*A complete listing of the SPI Research Group appears in part I of this study, which precedes this article (Mangano et al. [9]).

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temporal course of myocardial ischemia during the prolonged (1 week) postoperative period in at-risk patients undergoing noncardiac surgery.

Methods

Study subjects. We studied 100 men with or at risk for coronary artery disease scheduled for elective noncardiac surgery with general anesthesia at the Department of Veterans Affairs Medical Center in San Francisco. The study protocol was approved by our Committee on Human Research and all patients provided informed consent. The study group and inclusion and exclusion criteria are described in Part I of this report (9).

ECG measurements. Patients were monitored preoperatively to obtain control measurements, assess the effect of positional variation and provide baseline ST segment information needed to set ischemic threshold limits. Continuous postoperative monitoring began the 1st h after surgery and continued for up to 7 days. We used a single channel solid state electrocardiographic (ECG) monitor (QMED Monitor One Star), chest lead CM_5 (12) and silver/silver chloride electrodes with skin impedance <5 kΩ in all patients. The effects of position on the ECG morphology were measured using 1 min ECG samples obtained with the patient in the supine, left lateral decubitus, right lateral decubitus and upright positions. The most depressed ST segment apparent in these four positional tracings was chosen as the baseline ST segment (to minimize false positive changes), and the monitor's programmable ST segment change threshold was set at 1 mm (0.1 mV) below that baseline value.

The solid state monitor is an ambulatory device that continuously records and analyzes the ECG. The incoming signal is digitized at 256 Hz and is fed to a 65C02 microprocessor for analysis in which signal amplitude is compared with a fixed internal reference used for calibration. The frequency response is uniform from 0.05 to 40 Hz (13–15). The algorithm detects and validates the QRS complex as a region of sustained slope that meets criteria for duration, peak amplitude and a number of inflection points. Ectopic beats and noise are excluded by means of QRS width criteria (QRS < 120 ms) and slope change criteria (slope change <1 mm in 60 ms). Validated complexes are analyzed to determine the onset, peak, J point and J + 60 ms point. The ST segment level average is updated on a beat by beat basis, and amplitude data are quantified in 1 mm steps. An ischemic event is declared by the monitor when the ST segment descends 1 mm below the program threshold for ≥60 s. The duration of an ischemic event is measured from the time when the ST segment crosses threshold to a point approximately 40 s after return of the ST segment above the 1 mm threshold. The output of this monitor consists of tabular information describing a number of events, duration of events and duration at the 1, 2 and 3 mm thresholds over the entire time period. In addition to these quantified data, waveform data are stored in the solid state random access memory for subsequent retrieval. Up to 25 hard-copied 10 s samples of the ST ischemic events can be stored. Our data were downloaded daily and the 25 event limit was never exceeded.

We accepted only those reported ECG ischemic episodes that were accompanied by hard copy and validated by two independent investigators (D.M., J.T.) who were unaware of patient identity or clinical course; disagreements were resolved by consensus, involving a third investigator (M.L.) if necessary. Our criteria also demanded that ischemic events be reversible and that ST changes occurring in the presence of T wave changes (more nonspecific) have 1 mm depression from baseline at both the J + 60 ms *and* J point. For each ST episode, we measured: 1) onset time, 2) duration, 3) maximal ST change, 4) ST slope, 5) heart rate (mean value of the 15 min period preceding the episode and during the first min of the episode), and 6) symptoms.

Hemodynamic measurements. Heart rate was continuously recorded by the solid state monitor during the entire 8 day perioperative period. Heart rate reported at the time of the ischemic episode was confirmed using sample ECG strips. Other values for heart rate were extrapolated from the heart rate trend graphs.

Clinical care. Research data were collected in parallel to clinical data, and all physicians providing clinical care had no knowledge of any study data. Details of clinical care are described in Part I of this study (9).

Outcome measurements. The methodology for the outcome measurements is described in Part I (9).

Data analysis. Chi-square analysis with continuity correction was applied to categoric data. Student's *t* test was used to test the difference between the mean values in two groups. Multivariate analysis of variance using repeated measures was used to detect differences among periods (pre-, intra- and postoperative). If significant, pairwise comparison using Student's *t* test was used to determine differences among specific periods (16,17). Episode characteristics (such as duration) were compared over the three periods by first averaging the duration of all episodes for each patient for each period and then using multivariate analysis as described. When no episode occurred, a zero was assigned. A p value <0.05 (two-sided) identified statistically significant differences. Results are expressed as the mean ± 1 standard deviation unless otherwise indicated.

Results

Demographics. The demographic and clinical characteristics of the study patients are presented in Part I (Table 1) (9).

ECG findings. Patients were monitored with solid state ECG for an average of 6 ± 2.3 days (total monitoring time 10,445 h). A typical hard copy output of a single patient's normal baseline ECG CM_5 complexes on postoperative days 1 and 2 is shown in Figure 1. ST depression progressively

Table 1. General Postoperative Electrocardiographic Characteristics of 100 Patients

	DOS	POD 1	POD 2	POD 3	POD 4	POD 5	POD 6	POD 7	Total
Total hours monitored	800	2,018	1,773	1,534	1,286	1,162	1,053	819	10,445
No. of patients with ischemia	9	16	15	13	12	7	8	6	27
No. of ischemic episodes	23	87	65	109	48	44	44	17	437
Total ischemic minutes	801	2,553	4,878	5,221	1,960	696	1,779	770	18,658
Ischemic min/h monitored	1.00	1.27	2.75	3.40	1.52	0.60	1.69	0.94	1.79

DOS = Day of surgery; POD = postoperative day.

worsened on postoperative days 1 and 2 before the occurrence of myocardial infarction on postoperative day 4.

Twenty-seven patients (27%) developed 437 episodes of ECG ST segment changes suggestive of myocardial ischemia ("ischemic episodes"—see Limitations). On any given day during the first postoperative week, between 9% (day of surgery) and 19% (postoperative day 4) of patients exhibited an ischemic episode (Table 1). The total duration of ischemia in all patients over the 1st postoperative week equalled 18,658 min, or 1.8 min of ischemia/h monitored.

The overall pattern and severity of ischemia over the 8 day postoperative period are illustrated in Figure 2. The ischemic burden was highest on postoperative days 2 and 3 (2.8 and 3.4 min of ischemia/h monitored, respectively), exceeding that on the day of surgery (1 min of ischemia/h monitored). However, marked ischemia persisted to postoperative day 6, characterized by 1.7 min of ischemia/h monitored, and 59% of these late episodes were severe: maximal ST change ≥ −2 mm (Fig. 2). The specific episode characteristics (Table 2) suggest that most episodes occurred early (postoperative days 1 to 3) and that early episodes tended to be longer (but not significantly so as a result of the large standard deviations using multivariate analysis of variance). Eighty-four percent of episodes were clinically silent, unaccompanied by symptoms of angina, pulmonary congestion or syncope.

The distribution of postoperative tachycardia episodes is shown in Figure 3. Postoperative tachycardia was most common on postoperative days 1 and 2, then decreased during the late postoperative period: 57% of the ischemic episodes were associated with tachycardia (Table 2).

Relation of ischemia to risk factors and outcome. Ischemia during the 1st postoperative week was equally common among patients with definite coronary artery disease (27%) or at risk for coronary artery disease (27%). Patients undergoing major vascular surgery had the highest incidence of

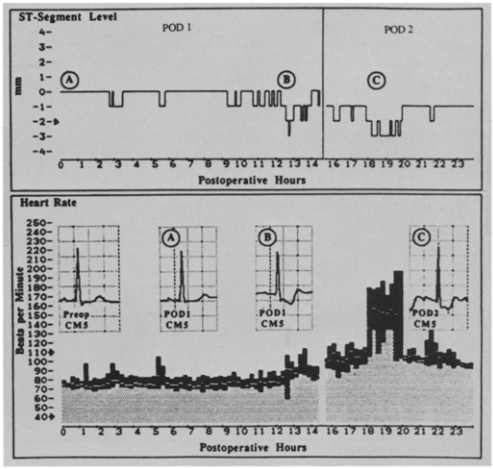

Figure 1. Solid state electrocardiographic (ECG) data from a patient who sustained a myocardial infarction on postoperative day (POD) 4. **Upper panel.** The ECG complexes from the preoperative control period (Preop), baseline (A) for postoperative day 1 (hours 0 to 14: midnight to 2 PM), a postoperative day 1 episode (B) and a postoperative day 2 (h 16 to 23: 4 to 11 PM) episode (C). **Lower panel.** Heart rate histograms for postoperative days 1 and 2. The threshold for ischemia and tachycardia are indicated by **arrowheads**. Because this patient had initial ST depression at J + 60 ms, the ischemic event threshold was set at −2 mm.

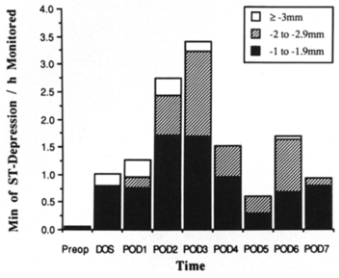

Figure 2. Pattern of postoperative ST depression. For each postoperative day (POD), the total minutes of ST depression were summed and then divided by the total duration of monitoring for all patients. The data were further divided into three groups: episode durations with maximal change from baseline −1 to −1.9 mm, −2 to −2.9 mm and ≥ −3 mm. For example, on postoperative day 6, patients had 1.7 ischemic min/h monitored, with episodes having a maximal change of ≥ −2 mm comprising 59% of the total ischemic minutes. Preop = preoperative.

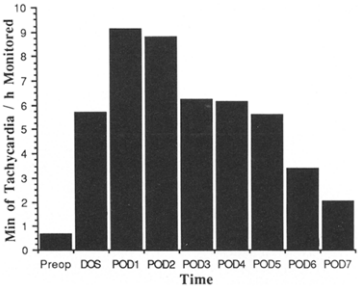

Figure 3. Pattern of postoperative tachycardia. For each postoperative day (POD), the average number of tachycardic (heart rate ≥ 100 beats/min) min/h monitored was calculated by dividing total minutes of tachycardia by total hours monitored. Preop = preoperative.

ischemia (38%) compared with those undergoing abdominal/thoracic (27%, p = 0.03) or other (orthopedic, neurosurgical, head/neck, peripheral vascular) (13%, p = 0.03) surgical procedures. There was no significant difference in the incidence of ischemia in patients receiving preoperative antianginal medication (nitrates, beta-adrenergic blockers, calcium channel blockers) versus those who did not (27% versus 26%) (chi-square = 0.02, df = 2, p = 0.9).

Thirteen (13%) of the 100 patients had an in-hospital cardiac outcome: fatal myocardial infarction in 1, nonfatal myocardial infarction in 3, unstable angina in 1, congestive heart failure in 4 and cases of ventricular tachycardia (≥5 consecutive beats) in 4. The overall incidence of postoperative ischemia was significantly higher in patients with an adverse outcome: 62% versus 22%, respectively (chi-square = 7.1, df = 2, p = 0.01), as was the incidence on each postoperative day (day of surgery 38% versus 5%; postoperative day 1 37% versus 10%; postoperative day 2 46% versus 10%; postoperative day 3 46% versus 8%; postoperative day 4 31% versus 9%; postoperative day 5 23% versus 5%; postoperative day 6 38% versus 3%; postoperative day 7 23% versus 3%). The total number of ischemic episodes during the postoperative period was also higher in patients with an adverse outcome: 15.9 versus 2.6 events per patient, respectively. All five patients who had cardiac death (n = 1), myocardial infarction (n = 3) or unstable angina (n = 1) during the 1st postoperative week had a postoperative ischemic episode ≥1 day before the outcome. Three of the remaining eight patients with congestive heart failure or ventricular tachycardia had preceding postoperative ischemic episodes.

Concordance between solid state and ambulatory ECG. In 88 of the 100 patients, we compared solid state ECG results

Table 2. Specific Electrocardiographic Episode Characteristics

	DOS	POD 1	POD 2	POD 3	POD 4	POD 5	POD 6	POD 7	Total
ST change (mm)									
Mean	−1.3 ± 1.0	−1.5 ± 0.7	−1.4 ± 0.9	−1.5 ± 0.7	−1.3 ± 0.7	−1.3 ± 0.5	−1.3 ± 0.5	−1.3 ± 0.5	—
Median	−1.0	−1.0	−1.0	−1.0	−1.0	−1.0	−1.0	−1.0	—
Duration (min)									
Mean	70 ± 76	85 ± 118	129 ± 144	130 ± 143	86 ± 136	67 ± 128	92 ± 119	112 ± 145	—
Median	25	25	73	50	15	15	27	33	—
Percent with symptoms	4	21	11	8	4	0	64	29	16
Percent with heart rate >100 beats/min	52	76	83	48	27	22	84	29	57

Abbreviations as in Table 1.

with ambulatory ECG results (9) during a 33 ± 11 h (postoperative day 1) period. (Twelve patients were excluded because of ECG lead differences or inadequate (<12 h) temporal overlap.) The concordance was 80% for this group of patients ($p < 0.01$, McNemar's test). In addition, a total of 338 ECG episodes were detected by either modality. Two hundred ninety-five (87%) of the episodes were similar in waveform (trend), complex and amplitude. Of the 43 episodes (13%) that differed, 21 (6%) had differences in R wave amplitude, 17 (5%) had differences in configuration (lead placement) and 5 (1%) had ST level differences.

Discussion

Our findings suggest that in at-risk patients undergoing noncardiac surgery: 1) postoperative ECG ST changes consistent with myocardial ischemia most often occur on the 2nd and 3rd days after surgery, but can persist for ≥1 week; 2) postoperative ischemia is clinically silent and therefore difficult to detect; and 3) postoperative ischemia may be related to a persistently elevated heart rate during the 1st week after surgery. Additionally, these preliminary outcome results suggest that both early and late postoperative ischemia may be associated with a severe adverse cardiac outcome.

Relation to previous studies. Other studies also have demonstrated that myocardial ischemia commonly occurs during the 1st 2 days after cardiac and noncardiac surgery. After cardiac surgery, we found a 42% incidence rate of postoperative ischemia in cardiac surgical patients (versus an 18% intraoperative incidence rate) (18), which was confirmed in an independent study (19) that found a 60% postoperative versus 4% intraoperative incidence rate of ischemia in similar patients. In at-risk patients undergoing noncardiac surgery, recent studies (1,9) have suggested that the incidence of early (up to 48 h) postoperative ischemia was also substantially higher (30% to 38% versus 14% to 21% intraoperatively). Moreover, both the magnitude and duration of early postoperative ischemia appear to be more severe. Knight et al. (18) also found that postoperative ischemic episodes were longer than intraoperative episodes in cardiac patients and that ST deviations from baseline values were more severe. We (20) recently confirmed these findings using both ECG and echocardiographic measures of ischemia. Similar findings also have been reported in noncardiac surgical patients (see Part I [9]) in whom the magnitude and duration of early postoperative ischemia appear to be significantly worse than in the preoperative or intraoperative periods. Finally, there is preliminary evidence in both cardiac (18,20) and noncardiac (1–5,10) surgical patients that postoperative ischemia detected by ECG or by echocardiography may have a strong association with adverse cardiac outcome.

Our findings complement and extend these results and demonstrate that silent postoperative ischemia can persist for ≥ 7 days after surgery, thereby confirming previous data suggesting that myocardial infarction occurs not only early but also late in the postoperative period, perhaps 3 to 7 days after surgery (2).

Potential mechanisms for postoperative ischemia. Multiple stresses occur during the early and late postoperative period and can substantially alter myocardial oxygen demand and supply and precipitate ischemia (11). Early postoperative stresses include: pain on emergence from anesthesia, fluid shifts, temperature changes, impaired pulmonary gas exchange and sleep deprivation (2,11). Late stresses may be associated with: ambulation, coagulation abnormalities, platelet activation with mediator release and changes in arachidonic acid metabolism. As a result, changes occur in adrenergic activity (sympathetic and parasympathetic) (11), plasma catecholamine levels (21–24), hemodynamics and ventricular function (25,26) and coagulation (2,11) throughout the in-hospital postoperative period. The persistently elevated heart rate noted throughout the postoperative period in our patients could have been a marker for any of these changes and may have been a mediator or a marker of myocardial ischemia. Few data, however, are available, necessitating studies addressing these potential mechanisms.

Silent postoperative ischemia. Postoperative ischemia was not only relatively common and persistent but also silent, making it difficult to detect using the usual clinical modalities. Eighty-four percent of all early and late postoperative episodes were clinically silent, unaccompanied by symptoms of angina, pulmonary congestion or syncope. These results complement our recent findings (9) using ambulatory ECG technology in these patients during the early postoperative period—94% of ambulatory ECG episodes were silent during periods when patients were able to communicate such symptoms. The mechanisms for the silent nature of postoperative ischemia are not known, but postoperative pain perception and differentiation may be altered after surgery because of residual anesthetic effects, administration of analgesic agents or competing somatic stimuli (such as incisional pain). Such abnormal somatic pain thresholds and altered pain perception have been observed in ambulatory patients with silent ischemia (27–30). However, it is noteworthy that silent ischemia was prevalent not only early but also late in the postoperative period, when pain perception and differentiation would have been expected to have normalized. Other still undiscovered mechanisms must also play a substantial role.

Other relations. Patients without coronary artery disease were as likely to develop postoperative ischemia as were those with coronary artery disease. Although surprising, this finding is consistent with our recent study (9) addressing early postoperative ischemia. One explanation is that the disease state in patients with coronary artery disease was stable without unstable angina, acute myocardial infarction or acute congestive heart failure. Therefore, differences between the actual presence of coronary artery disease and risk factors for coronary artery disease may be less impor-

tant than the effects of the *dynamic* physiologic changes that occur postoperatively.

Patients undergoing major vascular surgery had a higher incidence of prolonged postoperative ischemia, yet the incidence of immediate perioperative ischemia was no higher in these patients (9). Thus, the emergence from anesthesia combined with other stresses that occur in the early postoperative period (such as pain, hyperthermia) precipitate ischemia in a substantial number of patients (42%), regardless of the type of surgery. However, over the more prolonged postoperative period, the physiologic changes occurring in patients undergoing major vascular surgery appear to be more substantial, resulting in more morbid complications (2,4–12). Our findings are consistent with this. The 42 patients undergoing vascular surgery (versus the 58 undergoing nonvascular surgery) had greater postoperative fluid balances (intake-output 2,734 versus 2,021 ml, $p < 0.03$), more prolonged intubation and mechanical ventilation (13.3 versus 4.9 h, $p < 0.003$) and more prolonged intensive care unit stay (4.8 versus 1.7 days, $p < 0.0001$). Finally, in-hospital stay was substantially longer (19.3 versus 11.7 days, $p < 0.004$) in vascular versus nonvascular surgery patients.

Our results suggest that a relation may exist between postoperative ischemia and serious adverse cardiac outcome; thus, prolonged (1 week) postoperative monitoring and treatment of selected patients may be warranted. However, late (postoperative days 3 to 7) postoperative ischemia occurred in 12 of 22 patients with early (postoperative days 0 to 2) ischemia and in only 5 of 78 without early ischemia. Thus, the relative risk of late ischemia after early ischemia is 8.5, perhaps indicating that prolonged monitoring need be performed only in those patients who develop early ischemia (presuming ischemia is an important predictor of adverse outcome). However, such interpretation must be guarded because the number of outcomes was small; larger-scale studies are necessary to define the appropriate postoperative monitoring period.

Limitations. An absolute reference standard for myocardial ischemia does not exist and, even if available, would be difficult to apply because of the spontaneous occurrence of perioperative ischemia. Therefore, we cannot state whether the ECG ST segment abnormalities observed using solid state monitoring truly indicate myocardial ischemia. Changes in myocardial perfusion and ventricular function have been documented during episodes of silent ischemia (31–34). However, nonspecific ST segment changes may occur perioperatively as a result of changes in body temperature, serum electrolyte levels, ventilatory or positional changes or administration of drugs. Establishing the predictive importance of perioperative ST abnormalities may be the only method, albeit indirect, for assessing their "validity."

We used a solid state monitor for the detection of postoperative ischemic changes during the 1st week after surgery. We balanced the limitations of solid state monitoring against its distinct advantages (namely, that it readily allows analysis of large amounts of ST data collected continuously over prolonged periods). In our study, >10,000 h of solid state data were collected in 100 patients. Ambulatory ECG analysis would have required substantially more resources. We therefore chose to use solid state monitoring, offsetting several of its limitations by using conservative ST criteria and hard copy verification of all episodes by three investigators who had no knowledge of study data.

Several studies (13–15) have addressed validation of the solid state algorithm in detecting ST deviation. However, it is necessary to cautiously interpret solid state monitoring results because of its associated limitations. First, the solid state monitor allowed only single lead ECG detection. Use of a single ECG lead to detect myocardial ischemia is a limitation. We estimate that approximately 30% of ST changes were not detected. Our previous study (12) using continuous 12 lead ECG monitoring demonstrated that 75% of intraoperative ST changes are detected when a single modified V_5 lead is used. In addition, we did not measure ST elevation, which constitutes approximately 5% of ST changes (12).

Second, baseline selection is another important limitation of solid-state monitoring. The threshold for ischemia detection is programmed at the start of the monitoring period and usually is not readjusted (13–15). Two types of error can result. First, the ST baseline may drift downward during the monitoring period (usually 24 h) and this drift may not be due to ischemia but instead to other slowly changing processes (conduction, electrolyte or temperature changes). Thus, more "episodes" will be detected. Second, an initial baseline deviation may be abnormal (and in fact may represent ischemia), but may normalize over the monitoring period. However, the initial threshold will be set at the abnormal (low) ST level. The initial episode will not be detected; in addition, episodes that occur subsequently to normalization of the baseline may not be detected.

Third, the automated detection algorithm may incorrectly identify primary T wave changes as ST changes when they cause depression of the latter part of the ST segment. We designed our criteria to prevent this error.

Fourth, positional changes may cause ST changes. We accounted for this by making our criteria even more stringent, requiring ST changes to be ≥1 mm plus the maximal change induced by position. We chose to be conservative, improving specificity at the expense of sensitivity.

Fifth, because only a limited sample of the hard-copy ST data are provided by the solid-state monitor, the baseline (as already discussed), episode onset and offset characteristics cannot be validated. Similarly, detailed heart rate data are provided only at selected times; thus, the relation between heart rate and ischemic episodes can only be estimated.

Sixth, the monitor threshold can be set only in 1 mm increments. We chose to be conservative by setting the threshold at −2 mm if the baseline was between −0.1 and −0.9 mm, at −3 mm, if baseline equals −1 to −1.9 mm and so on. Again, we increased specificity at the expense of

sensitivity. In addition, because of this threshold limitation, episode characteristics (such as ST magnitude or duration) derived from solid state ECG cannot be directly compared with those obtained from other technologies that use a continuous threshold, such as ambulatory ECG (see Part I [9]).

Conclusions. In summary, in at-risk patients undergoing elective noncardiac surgery, postoperative myocardial ischemia can be prolonged, silent and therefore difficult to detect and related to a persistently elevated heart rate. Our data also suggest a relation to adverse outcome. If postoperative ischemia proves predictive of adverse outcome, extended monitoring and aggressive therapy for control of pain and heart rate may be warranted during the 1st week after surgery.

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